Idaho Department of Health and Welfare

Idaho Disease
Bulletin

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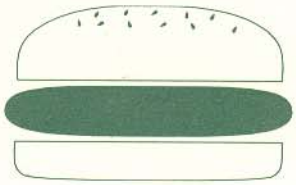
Hemolytic Uremic Syndrome
It's not just an E. coli O157:H7 problem.....



Hemolytic Uremic Syndrome (HUS):

HUS is a potentially life-threatening condition, which is known, in some cases, to be caused by *E. coli* O157:H7. HUS is the most common form of acquired renal insufficiency in infants and young children. In Idaho, cases of HUS have occurred due to E. coli O157:H7, and *E. coli* O26:H11; a non-O157:H7, toxin-producing *E. coli*.

Clinical Findings:

Usually there is a prodrome of hemorrhagic colitis 5-7 days prior to the onset of renal insufficiency. This may be followed by pallor, irritability, hypertension, hyponatremia, hyperkalemia, hypocalcemia, anemia, thrombocytopenia, metabolic acidosis, and seizures.

Acute renal failure is due to the effects of the liberated toxin (shiga toxin, verotoxin) rather than by immune-complex deposition. The toxin causes a break-down in prostacylin metabolism in endothelial cells, causing a disruption in renal capillary integrity. A second reason for renal dysfunction is vascular accumulation of cellular debris from hemolysis.

Serious complications leading to death have been documented in > 5% of HUS cases. Although most affected individuals survive, between 10% and 30% of survivors will have some level of permanent kidney damage.

Etiologic Agents:

Although *E. coli* O157:H7 has been the most well-documented cause of HUS to date, the liberated toxin has been found in several other enteric pathogens. When HUS is found and a standard lab examination for *E. coli* O157:H7 is negative, the identification of other agents should be attempted. These include non-O157:H7 shiga-toxin (verotoxin) producing *E. coli* (such as #O26, O111, O121, O145), Shigella, and Campylobacter.

Laboratory Testing:

Microbiology laboratories can screen for *E. coli* 0157:H7 by inoculating stool specimens onto sorbitol-MacConkey medium (SMAC plates). If this screening is negative for O157:H7 *E. coli*, further testing is often **not** done to find non-O157 *E. coli,* unless specifically requested by the physician. Cont'd→

INSIDE:

If the laboratory is made aware that HUS is occurring in the patient, further investigation into the less frequently identified microorganisms may be arranged.

Non-O157:H7 E. coli must be identified using methods available in reference laboratories, including the State Bureau of Laboratories in Boise. These methods detect either free fecal toxin or detect toxin genes using polymerase chain reaction (PCR) amplification.

Non-O157:H7 E. coli are important contributors to HUS and are usually not specifically identified without specific instructions to do so by the attending physician.

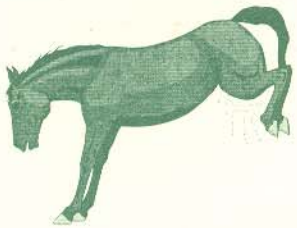
Prolonged excretion of *E. coli* O157:H7 in stools is uncommon, and the rate of recovery of *E. coli* O157:H7 from an infected individual's stools declines after the sixth day of illness. Therefore, stool samples should be obtained as early in the clinical illness as possible. A negative culture from a stool specimen obtained after the sixth day of illness or from stool obtained from a child receiving an antimicrobial agent does not exclude the possibility of *E. coli* O157:H7 infection. Furthermore, a negative culture for O157:H7 at any point does not exclude the possibility of infection by other toxin-producing enteric pathogens.

Reportability:

E. coli O157:H7 is currently reportable in the State of Idaho. Non-O157 shiga-toxin producing *E. coli* infections and HUS have been added to the list of Idaho Reportable Diseases and Conditions to become effective with the next revision of the Rules and Regulations Governing Idaho Reportable Diseases.

Rabid Horse in Eastern Idaho

In February, 1999, several farm workers were bitten and chased by a rabid horse on a ranch in Eastern Idaho. In addition, attending veterinarians were also exposed to body fluids and neural tissue before a diagnosis was conclusive for rabies.



Ultimately, seven individuals required rabies post-exposure prophylaxis as a public health precaution.

This is the first horse ever known to be diagnosed with rabies in Idaho; therefore, there wasn't a high level of suspicion for rabies when the horse became aggressive. The horse was infected with the bat strain of rabies, which is the only strain currently being identified in Idaho.

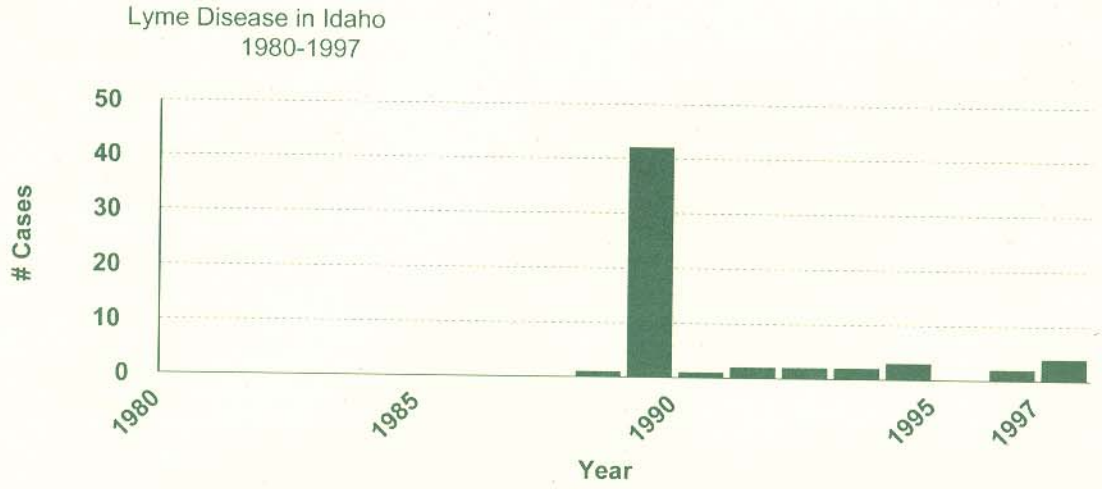
The presumed source of infection was a barn with roosting bats located in the horse pasture.
The nine remaining pasture mates are being quarantined on-site for 6 months and vaccinated against rabies.

Idaho Reports 13th

Case of Hantavirus Pulmonary Syndrome Since 1978

North-Central Idaho reported its first ever case of hanta-virus pulmonary syndrome. Sin Nombre virus was identified from a fatal case in May, 1999. Biological risk factors included smoking, asthma, chronic respiratory infections, and allergies. Environmental risk factors included several opportunities for rodent exposure. For a more thorough review of Hantavirus in Idaho see Idaho Disease Bulletin Issue Vol 6, #1, February 1999.

What about Lyme Disease in Idaho?



Lyme disease: caused by *Borrelia burgdorferi*, is reported an average of 2 times a year in Idaho with the exception of 1989, where 42 cases were reported; most from northern counties. Lyme disease was a newly reported syndrome at that time and the dramatic number of reported cases may have been attributed to incomplete laboratory confirmation or reporting based on clinical presentation alone.

A significant number of reported cases each year have a documented travel history to endemic areas nationwide. The remaining individuals profess no out-of-state travel, which raises the issue of whether Lyme disease is endemic in Idaho.

The tick vector traditionally associated with Lyme disease is *Ixodes pacificus*, which has never been identified in Idaho.

The Idaho Department of Health and Welfare is currently conducting a statewide tick survey, in association with the District Health Departments, to determine if *I. pacificus* exists in Idaho.

Diagnostics: Erythema migrans, the best clinical marker of Lyme disease, only occurs in 60% to 80% of patients. A local hypersensitivity reaction to a tick-bite may have a similar appearance but will not have central clearing and will not last for weeks, as the EM lesion does. Lyme disease is not laboratory-confirmed, according to the Centers for Disease Control and Prevention, unless a two-test approach is taken.

Two-test approach:

An enzyme immunoassay (EIA) or immuno-fluorescence antibody (IFA) test must be performed and confirmed by a follow-up Western blot.

The reason for a follow-up Western blot is because there is strong biologic cross-reactivity between all *Borreliae* that is non-discriminatory using the EIA or IFA test. This is important in Idaho as there is endemic tick-borne relapsing fever caused by *Borrelia hermsii*, which cross-reacts in the Lyme disease EIA or IFA. Relying on only the initial EIA or IFA test is not sufficient confirmation to diagnose a case with Lyme disease. The Western blot can usually sort out the differences between organisms by examining individual, species-specific proteins.

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